DELAWARE STATE MEDICAL JOURNAL

Issued Monthly Under the Supervision of the Publication Committee
Owned and Published by the Medical Society of Delaware

VOLUME 18
NUMBER 5

MAY, 1946

Per Copy, 20c
Per Year, $2.00

HEMOCHROMATOSIS*
Report Of Case With Necropsy

EDWARD M. BOHAN, M. D.,**
Wilmington, Del.

Hemochromatosis, or bronze diabetes, is a rare disease of metabolism, the cause of which is unknown. The liver is primarily affected with an abnormal deposition of iron containing pigment. The pancreas is affected early in the course of the disease and this accounts for the diabetic characteristics of the disorder, which are present in 80 to 90 per cent of cases. The lymph nodes, striated muscle, alveolar epithelium, reticulo-endothelial system and skin are subject to the pigmentation, although there are variations in the amount of pigment present. For example, the skin does not show pigmentation in 20 per cent of cases. Duncan[1] states that the disease is twenty times as common in males as females. In the Mayo Clinic, Wilder[2] reports that only 34 cases have been recorded over a period of 15 years. Berk and Lieber[3] performed autopsies on 12 cases at the Philadelphia General Hospital, from January, 1920, to December, 1937. 311 cases were all that could be collected by Sheldon[4] in his review of the subject. Humphrey, Alpiner and Verity[5] say that only 400 cases were reported in the literature until 1942.

The derivation of the pigment has resulted in many interesting opinions and discussions. Boyd[6] does not think that the pigment is derived from the blood because of the absence of hemolysis and hyperplasia of the bone marrow. Mallory[7] considers the metabolic disturbance to be related to chronic copper poisoning. He succeeded in producing the diseases in animals by the administration of copper. He thinks that the hemoglobin liberated from the wastage of red cells unites with the copper to form cuprohemol. The foreign metal is then freed and hemofuchsin formed. The latter is slowly changed into hemosiderin by enzymatic action. Rous and Oliver[8] produced a condition identical with hemochromatosis in rabbits by repeated transfusions of blood, so that large amounts of blood were constantly being destroyed. 20 to 50 grams of iron are present in the body as compared with three grams in the normal person. Berk and Lieber[3] say that an estimate of the time taken to accumulate this amount of iron is between 30 and 50 years before the diagnosis is made. In a statistical study Boulin[9] claimed that it may take 10 years or even the greater part of the patient's lifetime for the condition to progress, until the melanoderma, cirrhosis and diabetes mellitus are present as a triad of symptoms. The copper associated with the disease is increased four to five times above normal, and is present in nearly all the organs except the kidney.

Most writers on the subject seem to think that the disease is some inborn error of iron metabolism, so that the tissues cannot utilize the iron given to them, or that there is a defect in the excretion of iron taken into the body over a period of years. According to McCance and Widdowson [10] the iron content of the body is controlled through the regulation of absorption and not by excretion. Best and Taylor[11] state that iron is a one-way element, being excreted only in minute amounts. Iron is absorbed in the upper part of the small intestine and after absorption disappears from the blood being stored mainly in the liver, but also in the spleen and kidney. The acidity of the gastric juice and the need of the body for iron may regulate the absorption, continue Best and Taylor[11], in discussing the metabolism of iron. In one of the more modern theories, Butt and Wilder[12] think that the intestinal mucosa may be defective due to a deficiency in vitamin A. This is supported by the view of Flaum and Stoeck[13], who find the vitamin A content of the liver greatly reduced in hemochromatosis.

* Read before the Staff Meeting, St. Francis Hospital, May 7, 1946.
** Chief in Medicine; Associate in Metabolic Diseases, St. Francis Hospital.

The liver is changed to an orange brown color, but the color of the spleen and pancreas are unchanged. Shields Warren[14] in writing of the pathology of the disease says that the life of the cell is soon over as the pigment increases. Necrosis occurs and the stroma condenses. He claims that some attempt at regeneration takes place, considerable number of mitotic figures being found in the islets of the pancreas.

Malaise and asthensia, loss of weight and pigmentation are the earliest symptoms. Enlargement of the liver and diabetes mellitus are later symptoms. The secondary sex characteristics tend to disappear in many cases and according to Kelper et. al[15], this sign may appear early in the disease and be of diagnostic value. Morrione[16] thinks that the inability of the liver to inactivate estrogen may be responsible for the change in sex. Often the symptoms of diabetes are the first to be detected, even coma may have taken place. Anemia of a hypochromic variety appears. The blood sugar is elevated in many cases and the serum albumin and globulin ratio is reversed. If the diagnosis is not made by the foregoing symptoms and signs, a biopsy of the skin, analysis of the ascitic fluid, or urinary sediment may aid the clinician in his deductions. Beardwood and Rouse[17] describe a method by Fishback for the clinical demonstration of iron, in which an intradermal test is done with potassium ferrocyanide and hydrochloric acid. A positive reaction is present if a light blue color appears immediately and darkens to a deep blue within an hour. There also appears a peripheral red zone. A negative reaction is a white wheal within the peripheral red zone. Both of these reactions may take two weeks to fade away.

Brunsting[18] refers to Addison's disease chronic arsenic poisoning, and argyrosis in discussing differential diagnosis. The Addisonian syndrome is associated with an uniformly low blood pressure. The usual absence of diabetes and skin tests serve to distinguish the disease from hemochromatosis. Chronic arsenism gives rise to alternating areas of hyper- and depigmentation, creating a raindrop effect. It is also quite distinctive histologically. Argyrosis has a pigmentation which is more dark gray in color than brown. Glistening particles of silver will be revealed on dark-field illumination.

The cause of death is given in percentage by Duncan[1]. Diabetic coma, 50%; cirrhosis of liver, 11%; pneumonia, 10%; tuberculosis, 9%; and carcinoma of liver, 7%. The disease is invariably fatal, but the patients may be kept more comfortable by control of the complications that are present. Insulin resistance is common. 1,000 or more units of insulin have been given to cases in coma without result. This insensitivity to insulin is probably due to loss of storage capacity in the damaged liver for glycogen.

The duration of life previously assumed to be about a year and a half after diagnosis is made, has been appreciably prolonged by the discovery of insulin, the development of long acting insulins, and the earlier diagnosis and superior treatment of diabetes mellitus.

CASE HISTORY

The patient was a white male, aged fifty-five years. He was 5 feet, 10 inches tall, and weighed 145 pounds. In May, 1945, he noticed that a small inguinal hernia which had been present for years was protruding, so he decided to have it repaired. At operation the hernial sac was opened and the abdomen found to be filled with fluid. No further surgery was done and he was allowed to recover. He became well enough to spend the rest of the summer on vacation, returning to his home in September. The ascites grew gradually more severe. He was tapped at intervals of 10-14 days. Weight loss was quite apparent at this time. The onset of coma on October 27, 1945, resulted in his admission to St. Francis Hospital on October 28. The chief complaints were extreme weakness, drowsiness, loss of weight, and swelling of the abdomen. History of illnesses in the past revealed nothing of note, except for the fact that he had not been in robust health for seven or eight years. In other words he had been "ailing." Family history: Mother died of heart disease. Father died of intestinal obstruction. One brother and three sisters alive and well.

The physical examination revealed a fairly well nourished man, in no apparent distress. His face was flushed, he was drowsy, and acetone was present on his breath. The abdo-

men was markedly distended with fluid. There was a faint brownish pigmentation on the back, especially around the scapulae. On the dorsum of the fourth toe, on the right foot, there was a marked deep bronze pigmentation, which faded into a lighter pigmentation, as it approached the ankle. Two toes on the other foot were also deeply bronzed and there was a patchy lighter pigmentation on the tibial surfaces. The lower abdomen and forehead had a faint almost indiscernible spotty light brown color.

A blood sugar taken shortly after admission to the hospital was 323 mgm. per 100 cc. of blood. Later the blood urea was found to be 17 mgm., and the creatinine 1.5 mgm. Carbon dioxide combining power amounted to 24 volumes per cent. Plasma chloride was 370 mgm. The white cell count was 11,000; polymorphonuclears, 82%; lymphocytes, 15%; monocytes, 2%; and eosinophiles, 1%. The red cell count was 4,240,000, with 75% hemoglobin.

Treatment was instituted for diabetic acidosis with glucose, unmodified insulin, salt, and alkali. Salyrgan was administered intramuscularly daily for the ascites. Improvement was evident on the 30th of October, and the insulin was changed to 20 units of globin insulin, after the morning and evening meals. He also received amino acids, crude liver extract, and vitamins A and B complex. Diet was high in protein and carbohydrate.

The heart and lungs were found to be normal. Neither the liver nor the spleen could be felt. The diabetes became stable. The clinical symptoms and signs of the disease were not apparent. The insulin was gradually reduced to 14 units of globin in the morning and eight at night. This was evidence that the glycogen reserve of the liver was being added to. The theory of compatible hyperglycemia of Sindoni[19] was followed in treatment. The blood sugar level was kept between 170 and 220 mgm. All sugars were taken two hours after breakfast. Further readings were 196 on November 9, 170 on November 12, and 158 on November 20. The serum protein was 8, albumin 2, and globulin 6. The patient was comfortable and sat out of bed on occasions. The pigmentation gradually increased. No diminution in the ascites took place and the output never exceeded the intake, despite the daily doses of salyrgan in 2 cc. amounts.

The temperature was never over 99 degrees, except on the 19th of November, when it reached 100.2 degrees. The pulse stayed between 80 and 90 per minute. The patient remained very cheerful at all times during his illness. On November 25 he became restless and irrational. He continued this way off and on, until November 27, when abdominal pain seized him. At 11:20 p. m. his pulse became imperceptible and his respirations shallow and rapid. The abdomen was rigid and tender in the epigastrium. The patient expired at 8:10 a. m. the next morning.

NECROPSY REPORT*

The body is that of a well developed and fairly well nourished adult white male. There is no rigor mortis. The skin is slightly jaundiced and there are pigmented areas on the anterior surfaces of the lower legs, the dorsum of the hands, and upper chest. The abdomen is moderately distended and presents the marks of numerous puncture wounds. The feet and ankles are slightly edematous and there is a longitudinal scar on the dorsum of the right foot. The fourth finger on the right is missing. Eyes, ears, nose and mouth are not remarkable. Pupils are round, regular, and equal, each measuring 0.5 cm. in diameter.

Peritoneal Cavity: Surfaces are smooth and glistening. There are no adhesions. A large amount of thin watery fluid is present. This fluid pushes the diaphragm upward, but otherwise the abdominal organs are normally arranged. The mesenteric lymph nodes are not enlarged.

Pleural Cavities: Surfaces on the left are smooth and glistening. About 400 cc. of thin watery fluid are present. The pleural surfaces on the right are entirely covered with dense fibrous adhesions.

Pericardial Cavity: Surfaces smooth and glistening. There are no adhesions or excess of free fluid.

Heart: 340 grams. Normal in size, shape and position. Dissection of the organ shows no evidence of pathology.

* By Dr. Douglas M. Gay, Pathologist, St. Francis Hospital.

Lungs: The left weighs 320 and the right 550 grams. Both lungs are soft and crepitant throughout and the increased weight of the right lung is due to numerous fibrous adhesions which necessitated stripping off the parietal pleura in removing the lungs.

Spleen: 580 grams. About five times normal size. The capsule is irregularly thickened over a dark red surface. The splenic pulp is firm in consistence and cuts with slight resistance. It is dull red in color and very little pulp scrapes away.

Pancreas: 180 grams. About 50 per cent heavier than normal. It is firm in consistence and shows no definite gross pathology. A branch of the celiac axis running along one edge of the pancreas and probably representing the splenic artery is partly occluded with adherent gray thrombus.

Liver: 750 grams. About one-half normal weight but appears to be about one-third or one-fourth normal size. The organ is uniformly nodular throughout, the nodules being less than 1 cm. in diameter. The nodules are red-brown in color and are separated by an abundance of fibrous tissue, which gives the liver a firm consistence. The gallbladder is slightly thick walled and contains a few small dark brown calculi.

Adrenals: Normal in size, shape, color and consistence.

Urinary Organs: The combined weight of the kidneys is 370 grams. They are similar and are normal in size, shape, color and consistence. The capsule is thin and strips readily from a normal red-gray surface. The cortex averages 0.7 cm. in thickness. Calices, pelves, ureters, and bladder are normal.

Genital Organs: Grossly normal.

Blood vessels: The aorta is smooth and elastic in its upper portion and contains a few slightly raised plaques in the lumbar portion. The celiac axis is partly occluded by an adherent thrombus. The portal vein near the liver is also partly occluded by an adherent gray thrombus. Throughout the abdominal cavity there are numerous veins not ordinarily seen in the course of an autopsy that were made prominent as the result of varicosities and because of the development of collateral circulation. No superficial prominence of blood vessels is noted, but varicosities are present in the esophagus as noted below.

Gastro-Intestinal Tract: The veins of the esophagus are unusually prominent and by transillumination are seen to be distended tortuous structures. Portions of the surface are slightly roughened, but no definite perforations or ulcerations are seen. The stomach is distended with a large amount of blood, some of which is in the form of blood clot of recent origin and more of which is brown in color due to digestion. It is presumed that the fatal hemorrhage into the stomach originates from one of the esophageal varices, since there is no ulceration of the gastric mucosa and transillumination of this organ shows no evidence of varicose veins.

MICROSCOPIC

Heart: Normal.

Lung: Section includes a portion of the pleura thickened with scar tissue. The scar tissue extends into the adjacent lung tissue where a number of the alveoli are obliterated. Elsewhere, the alveoli contain edema fluid.

Spleen: There is an increased amount of fibrous tissue throughout and numerous monocytes filled with blood pigment are scattered through the pulp.

Pancreas There is diffuse fibrosis throughout the organ with obliteration of many of the islands of Langerhans. Throughout the fibrous tissue there are scattered monocytes filled with orange-brown pigment.

Liver: The portal spaces are enlarged with masses of scar tissue and lymphocytic infiltration. Strands of scar tissue extend from one portal space to others, subdividing the organ into a number of small nodules. The liver cells in the portal spaces contain prominent orange-brown granules. In the region of the hepatic vein, there is active destruction of liver cells with the necrotic cells undergoing phagocytosis by polymorphonuclear leukocytes. Other liver cells in this vicinity contain vacuoles within the nuclei. These vacuoles are interpreted as glycogen and indicate hyperglycemia at the time of death. Special micro-chemical reactions on the liver and all of the other organs in the autopsy indicate that the orange-brown pigment is blood pigment, thus proving the diagnosis of hemochromatosis.

Adrenal: The deep layer of cortical cells contains an abundance of orange-brown pigment.

Kidney: Essentially normal.

Skin: There is no definite abnormal pigment around the sweat glands or elsewhere.

Esophagus: Sections include several widely dilated veins, indicating varicosities.

SUMMARY

A case of hemochromatosis has been presented with three of the classical symptoms and signs, pigmentation, ascites and diabetes mellitus. The diabetes was controlled by dietary means and globin insulin. The ascites and pigmentation increased. The patient died from rupture of an esophageal varix.

CONCLUSIONS

1. Hemochromatosis is a rare disease. Etiology and pathogenesis are obscure.

2. Pigmentation may be absent so all cases of diabetes and ascites should be examined for hemochromatosis.

3. Diagnosis may be made by symptoms and signs, supplemented with laboratory tests of the blood, ascitic fluid, urinary sediment and skin biopsy. The intradermal test of Fishback[17] may replace the skin biopsy.

4. The diabetes and cirrhosis of the liver can in most cases be controlled by medical and surgical means adding to the comfort of the patient and prolonging life.

5. The earlier the diagnosis is made, the better the prognosis will be, because of the control of the diabetes. When the disease has progressed to the stage of severe liver damage the prognosis becomes more unfavorable.

6. The urine need not be used as a guide in diabetic coma. Observation of the patient, blood sugar readings and carbon dioxide combining power estimations may serve to control the acidosis or coma. Sindoni[20] advised this method of treatment in 1939. He found that no apparent correspondence existed between the blood sugar and urine sugar in a large number of patients with and without insulin.

608 W. 11th St.

REFERENCES

1. Duncan, G. G.: Diseases of Metabolism, 857. Philadelphia, 1942.
2. Wilder, R. M.: Clinical Diabetes Mellitus and Hyperinsulinism, 316. Philadelphia, 1941.
3. Berk, J. E., and Lieber, M. M.: Primary Carcinoma of Liver in Hemochromatosis, Amer. Jour. Med. Sci., 708-714, (Nov.) 1941.
4. Sheldon, J. H.: Lancet, 2:1031, 1934.
5. Humphrey, A. A., Alpiner, S., and Verity, L. E.: Hemocromatosis: Report of Case and Comments on Methods of Making a Definite Diagnosis. Arch. Derm. and Syph., 45:1128 (June) 1942.
6. Boyd, W.: Pathology of Internal Diseases, 4th Ed. 376-377. Philadelphia, 1945.
7. Mallory, R. B.: Amer. Jour. Path., 1:117, 1925.
8. Rous and Oliver: Osler's Practice of Medicine, 10th Ed., 448.
9. Boulin, R.: Statistical Study of 70 Cases of Bronze Diabetes, Presse Medicale, 53:326 (June) 1945.
10. McCance, R. A., and Widdowson, E. M.: Lancet 2:680, 1937.
11. Best, C. H., and Taylor, H. B.: App. Phys., 3d Ed., 97.
12. Butt, H. R., and Wilder, R. M.: Hemochromatosis: Report of 30 Cases in Which Diagnosis Was Made During Life. Arch. Path., 26:262 (July) 1938.
13. Flaum, G., and Stoeck, J. H., Jr.: Report of Case of Hemochromatosis With Necropsy and Analysis of Liver. Arch. Int. Med., 63:433 (March) 1939.
14. Warren, S.: The Pathology of Diabetes Mellitus, 183. Philadelphia, 1938.
15. Kelper, E. J., Nichols, D. R., and Mills, J. H.: Diagnostic Value of Regression of Secondary Sexual Characteristics in Cases of Hemochromatosis, Arch. Int. Med., 14:810 (Nov.) 1940.
16. Morrione, T. G.: Effects of Estrogens on the Testes in Hepatic Insufficiency, Arch. Path., 37:39 (Jan.) 1944.
17. Beardwood, J. T., Jr., and Rouse, G. P., Jr.: Year Book of General Medicine, 675. Chicago, 1945.
18. Brunsting, L. A.: Cutaneous Manifestations of Systemic Disease, Med. Clin. N. A., 863, (May) 1937.
19. Sindoni, A., Jr.: Personal Communication.
20. Sindoni, A., Jr.: J. A. M. A. 112:2595-2600 (June 24) 1939.

PERITONITIS OF APPENDICEAL ORIGIN *

G. S. SERINO, M. D.,**
Wilmington, Del.

Appendicitis is still a highly important problem because of the great frequency of the condition, and because it continues to be responsible for many preventable deaths. The mortality rates in first-class hospitals have been gradually improving, this, however, does not reflect the state of affairs in the population at large. According to Sperling and Myrick[1], appendicitis accounts for 20,000 deaths annually in the United States, the highest mortality rate in any civilized nation in the world. The fact that each year over 10 per 100,000 population die of appendicitis is regrettable, because appendicitis should be associated with little or no mortality, if the diagnosis is made and the proper therapy instituted while the infection is still confined to the appendix. It is only when the infection has extended beyond the appendix, either as a result of catharsis or early necrosis because of ischemic gangrene, that fatal complications occur. It is because of the still relatively high incidence of these complications that a study of appendiceal peritonitis is presented.

Up to the present time we have treated surgically 100 patients with peritonitis of appendiceal origin, without a death. It is because

* From the Department of Surgery, St. Francis Hospital.
** Chief in Surgery, St. Francis Hospital.

of the definite reduction in mortality and in order to evaluate the results which have been obtained that we are prompted to discuss some of our personal experiences.

This study includes all patients with peritonitis of appendiceal origin which were operated upon during the past 5 years. The ages ranged from 3 to 68 years. The duration of symptoms varied from 24 hours to 10 days. Two patients were admitted with peritonitis and pneumonia. Laxatives were taken by 14 patients. One patient in this group took two laxatives, caster oil in the morning and epsom salts in the afternoon. Much has been said about the danger of purgatives in patients with acute appendicitis. There is adequate evidence to show that caster oil or other active laxatives will greatly increase intestinal and appendiceal peristalsis so that perforation and dissemination of infection are more likely to occur.

In the natural course of the disease, checking of the inflammation will depend upon the immunity of the individual and upon the availability of mobile structures for walling off the process. The complete surrounding and enclosure of the appendix in a protective omental sheath takes place in some cases. A less effective mode of defense occurs when loops of intestines, bladder, mesentery or peritoneum become adherent to one another which enclose the appendix and the pus which surround it. If these walling off reactions fail to limit the infection, the general peritoneal cavity becomes contaminated.

We have made no attempt to distinguish between "local," "spreading," and "generalized" peritonitis. This is in agreement with Ladd[2], who states that "No surgeon really knows how diffuse the process is unless he has done a very improper operation."

Material for culture was taken in a routine manner at operation in each instance. The bacteriology in peritonitis is variable. In this series the following identifications were made: colon bacillus, 62; mixed growth, 30; streptococcus nonhaemolyticus, 1; streptococcus haemolyticus, 4; streptococcus aureus, 4.

It is of interest to note that three of the cases which were positive for mixed groups had bowel perforation. In two cases the appendix was "blown out" at its cecal attachment, with gross fecal contamination of the peritoneal cavity. In the other instance a perforation was present in an adjacent loop of small bowel.

From a laboratory standpoint the leukocyte count in 9 of these cases was normal. In the 91 other cases the count varied from 11,000 to 25,000. The polymorphoneulear average count was 82%.

There is a good deal of controversy concerning the treatment of appendiceal peritonitis. There is a divergence of opinion concerning when to operate, how to operate, whether to drain, and what the after care should be. In our opinion most cases of generalized peritonitis run a smoother course, have a better chance of recovery and have a shorter hospitalization if the appendix is removed and modern postoperative care instituted. This implies that appendectomy must be performed with a minimum of trauma, with little or no disturbance of other intraperitoneal structures. In general we conform to the policy of appendectomy and drainage for all cases with peritonitis. The very toxic cases are given adequate preoperative preparation. In some clinics a definite symptom duration, such as 48 or 60 hours, is used to decide the question of immediate operation. We do not agree with this arbitrary method of dictating forms of treatment, because some patients may still be in good general condition 60 hours after the onset of symptoms, whereas others have a ruptured appendix and spreading peritonitis in less than 12 hours. Therefore, we consider each case individually and the mode of treatment is based upon the actual condition of each patient as observed on admission. We are also convinced that it is wiser not to remove the appendix in cases of appendical peritonitis if adhesions must be broken down in its removal. This is particularly true if the process is well walled off. When laparotomy reveals an open perforation through which fecal material continues to discharge, appendectomy aids the defensive mechanism of the body by preventing further peritoneal soiling, but when the perforation and the appendix have been sealed off by surrounding intestine and omentum, removal of the appendix, by breaking down the already established defensive

measures of the body, may do more harm than good.

If there is any doubt about perforation we do an exploratory laparotomy, for we feel that the damage done by exploratory laparotomy, with or without appendectomy, is far less than that incurred by allowing an unruptured appendix to go on to perforation. The majority of patients with generalized peritonitis of appendical origin exhibit generalized abdominal pain and tenderness, absence of peristalsis on auscultation, rebound tenderness referred to the point of palpation of the left side of the abdomen, tenderness on both sides on rectal or vaginal examination, and distention. In some patients, however, particularly after localization has begun, one or more of these may be absent and the diagnosis of appendical peritonitis may be extremely difficult to make except by exploratory laparotomy. The converse may occasionally be true, for a patient whose appendix has not ruptured may present signs and symptoms suggestive of appendical peritonitis. This has been observed particularly of patients entering the hospital within 24 hours after the onset of their disease, and we have come to feel that the patient with acute appendicitis who enters the hospital within 24 hours of the onset is better treated by exploratory laparotomy and, almost without exception, removal of the appendix, regardless of the presence or absence of the clinical signs of perforation and generalized peritonitis.

In those patients with acute appendicitis in whom distention and generalized abdominal tenderness develop within 24 hours, the symptoms in my experience have been due more frequently to peritoneal exudate and vigorous catharsis than to gross perforation of the appendix. Another reason for urging appendectomy for these patients is the fact that if the appendix becomes ruptured within 24 hours the peritoneal cavity has had little time to combat such gross contamination and is poorly prepared to localize the infection. Operation in these circumstances usually shows an open perforation which continues to discharge fecal material, in contradistinction to those occurring from 72 to 96 hours after the onset of symptoms, which are usually largely sealed by omentum or surrounding intestine.

Two patients in this series entered the hospital with a diagnosis of peritonitis of appendical origin. Clinical and x-ray studies on these patients prior to operation showed the presence of pneumonia and peritonitis. Both patients had their appendectomy performed under continuous spinal anesthesia, as advocated by Lemmon[3]. The progress of these patients is shown in Figs. 1 and 2.

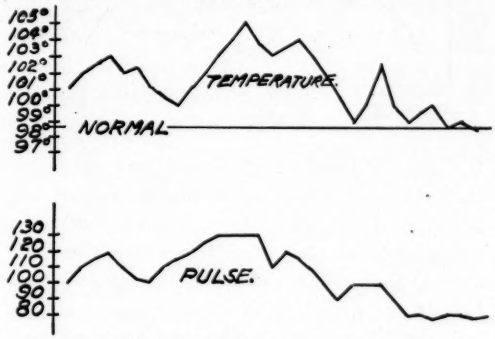


Fig. I—Clinical chart of patient with Peritonitis and Pneumonia.

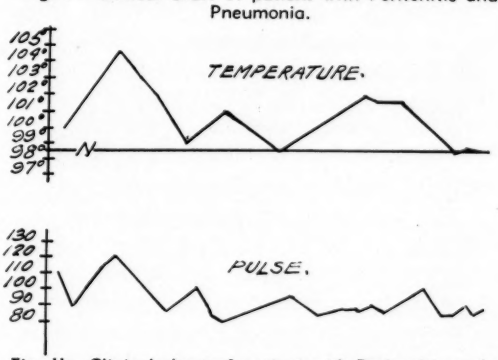


Fig. II—Clinical chart of patient with Peritonitis and Pneumonia.

A constant search for intraperitoneal abscesses is a part of the treatment of appendical peritonitis. While these may form in many portions of the abdomen and pelvis the most serious and the most difficult to detect are those formed in the subphrenic space. The mortality following the formation of a subphrenic abscess has always been very high, the greater number being found on the right side. Fortunately, only a small number of cases develop a left subphrenic abscess, and the mortality rate from this complication is extremely high. One patient, a boy of 8 years, developed a left subphrenic abscess which re-

quired a subcostal extraperitoneal incision and drainage. The convalescence and outcome is shown in Fig. 3.

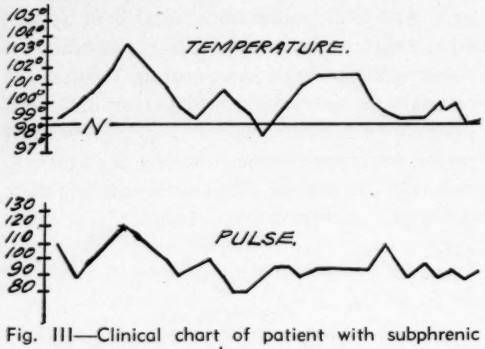


Fig. III—Clinical chart of patient with subphrenic abscess.

The appropriate treatment of secondary intraperitoneal absceseses is highly important. Many of these secondary abscesses will subside spontaneously. This has been my experience in the majority of cases. Delay in operation is our choice, with incision and drainage only in those cases presenting a severe toxemia with a rising pulse and increasing fever.

The postoperative treatment of these cases was definite, and was directed toward increasing the immunity of the peritoneum, improving the patient's resistance, preventing and relieving distention, diminishing intestinal activity, and localizing the disease.

The position of the patient in bed is very important. All of our cases were placed in Fowlers' position. This aids in the localization of the infection in the pelvis, where abscesses are more easily detected and drained. Breathing is made easier in this position, and the vital capacity is also increased. Breathing exercises are always used in these patients.

We apply heat to the abdomen in the majority of such cases. According to Oschner[4], heat tends to decrease the incidence of ileus. Most patients feel more comfortable with the application of heat to the abdomen. Warm stupes are used rarely.

The use of decompression by means of an indwelling duodenal tube with suction, such as the Miller-Abbott tube or the more recent Harris tube, is used in every case. This provides physiologic rest for the gastro-intestinal tract. Decompression empties the bowel of secretions and gas until normal peristalsis returns. This is the most effective method of treating the vomiting and distension associated with peritoneal infection.

Maintenance of a proper fluid, electrolyte, protein and vitamin balance is an individual problem. Blood and plasma transfusions were administered to meet individual requirements in which anemia and hypoproteinemia were factors. Five per cent glucose solutions in normal saline, alternating with distilled water, was utilized by the continuous drip method, giving nothing by mouth in the severe cases.

Morphine was routinely administered. Orr[5] concludes that morphine stimulates rhythmic contractions of intestinal muscle and raises the muscle tone as evidenced by increased intraluminal pressure. However, this same author[6] in a later report concludes that morphine may be given with assurance in the treatment of peritonitis not only to increase muscle tone but to make the patient comfortable, promote rest, relieve anxiety, and minimize thirst.

Oxytocic drugs such as prostigmine, pitressin, etc., were avoided. We believe this therapy might prove harmful in the treatment of ileus associated with peritonitis. The utilization of decompression seems more desirable under these circumstances with less possibility of bowel damage and spread of infection.

Clinical and experimental studies during the past several years have demonstrated the value of sulfonamides in the treatment of appendiceal peritonitis. In 1943[7] we reported a group of cases showing the beneficial effects of sulfathiazole. We have continued to use sodium sulfathiazole intravenously in many of these cases. For several years the local intraperitoneal implantation of sulfanilamide was used, but more recently we have discontinued this practice. There is no doubt, however, that sulfathiazole by the intravenous route has definitely contributed to the success of these operations.

During the past year we have been using penicillin in the majority of cases, either alone or in combination with sulfathiazole. Very little is known concerning the therapeutic possibilities of large doses of penicil-

lin. Laboratory experiments indicate that the E. coli is not susceptible to treatment with penicillin, but clinical evidence is accumulating showing that large doses are effective in controlling mixed infections. By controlling these other organisms penicillin may control peritonitis or keep the infection from spreading. The usual therapeutic dose of 25,000 to 50,000 units of penicillin is inadequate in the treatment of peritonitis. Our usual procedure is a starting dose of 100,000 units, reducing the dosage in accordance with the clinical improvement of the patient. This high initial dosage is important in mixed infections so that the penicillin-neutralizing action of the E. coli can be overcome.

The slow reduction of penicillin is also important because if discontinued abruptly the infection usually recurs. Large doses may prevent the development of secondary localized intraperitoneal abscesses. The intramuscular route was used in the majority of my cases, but the intravenous route was used in the very toxic cases. In a recent study Green and Alture-Weber[8] found that the intraperitoneal installation of penicillin was followed by significant bacteriostatic concentrations of the antibiotic in the blood. They concluded that penicillin was of prophylactic value against infections of abdominal origin.

Shafiroff[9] in a more recent article studied the rate of absorption of penicillin through the peritoneum into the blood stream in 22 normal patients. Penicillin appeared in significant quantities in the blood for a period of about five hours after its instillation in the abdomen.

Intraperitoneal implantation of penicillin was not utilized in this series. That the mortality and morbidity rate might still be more favorably influenced by this procedure is still in doubt. Our results during the past year have clearly demonstrated the value of penicillin by the intravenous or intramuscular route as an adjuvant to surgery in the treatment of appendiceal peritonitis.

A significant feature of modern surgery is the widespread interest and activity displayed in the investigation of the causes of postoperative morbidity and mortality. As a result, new principles, methods, and therapeutic suggestions have arisen. We have followed the suggestions that influence the operative mortality and the postoperative morbidity. Today the uncertainty of surgery in appendiceal peritonitis has been changed to an operative procedure with a much greater margin of safety.

There is no more distressing situation in surgery than that associated with peritonitis of appendiceal origin. There is no result in surgery which is more gratifying than the successful relief of this condition, but success calls for good surgical judgment and technical skill.

CONCLUSIONS

1. A series of 100 cases of peritonitis of appendiceal origin, without a mortality, is presented.
2. Sound surgical judgment and technical skill are important factors in the management of peritonitis.
3. Surgery combined with chemotherapy has reduced the mortality and morbidity.
4. Rigid adherence to surgical principles must be followed when chemotherapy is employed.

REFERENCES

1. Sperling, L., and Myrick, J. G.: Acute Appendicitis; A Review of 518 Cases of the University of Minnesota Hospital From 1932 to 1935, 1:255, 1937.
2. Ladd, W. E.: Immediate or Deferred Surgery for General Peritonitis Associated With Appendicitis in Children, J. A. M. A., 115:1239, 1940.
3. Lemmon, W. T.: A Method for Continuous Spinal Anesthesia, Ann. Surg., 111:141, 1940.
4. Oschner, A. J.: The Cause of Diffuse Peritonitis Complicating Appendicitis, and Its Prevention, Am. J. Surf. & Gyne., 15-84, 1902.
5. Orr, T. G.: The Action of Morphine on the Small Bowl and Its Clinical Application in the Treatment of Peritonitis and Intestinal Obstruction, Ann. Surg., 98:835, 1933.
6. Orr, T. G.: Treatment of Peritonitis, J. A. M. A., 113: 1489, 1939.
7. Serino, G. S.: Sulfathiazole Therapy in General Surgery, Am. J. Surg., 61:400, 1943.
8. Greene, H. J., and Alture-Weber, E.: Penicillin as a Prophylactic in Abdominal Surgery, Proc. Soc. Exper. Biol. & Med., 58:211, 1945.
9. Shafiroff, B. G. P.: The Peritoneal Absorption of Penicillin, Surgery, 18:753, 1945.

TREATMENT OF AN OBSTINATE CASE OF AEROBACTER BACILLURIA WITH STREPTOMYCIN *

B. B. G. BLACKSTONE, M. D., M. D. SOMMERNESS, M. D., and E. G. SCOTT, M. T., Wilmington, Del.

The introduction of a new antibiotic, streptomycin, which has proved effective in vitro against certain pathogenic gram-negative rods, has only recently been followed by clinical trial. The total published clinical data,

* From the Departments of Medicine and Pathology, the Delaware Hospital.

at the time of writing, on the use of streptomycin in infections (other than tuberculosis) has been recently increased to 82 cases, with reports of 45 by Herrell and Nichols[1]; 30 by Reimann, et. al[2]; and 7 by Foshay[3].

In the experience of two of these authors, streptomycin proved effective in the treatment of urinary tract infections caused by susceptible strains of some gram-negative rods. Herrell[1] reports results which he considered good in 10 of 13 cases of this type of infection, but commented that "best results are most likely to be obtained when the organism of infection is either *Proteus ammoniae* or *Aerobacter aerogenes.*" Reimann[2] reports that "streptomycin in adequate dosage promptly suppressed or eliminated *B. pyocyaneus, B. proteus morganii* and *E. coli* from the urine of several patients but only when these bacteria were of strains susceptible to the amounts of streptomycin present in the urine."

Since the evaluation of any new drug must await extensive clinical trial, the following case is presented as an addition to the above series.

CASE REPORT

A 77-year-old white, male patient first sought medical attention in June, 1945, at which time he underwent a suprapubic prostatectomy for relief of urinary frequency and retention. Postoperative recovery and convalescence was apparently uneventful.

The first admission to the Delaware Hospital was on September 18, 1945, for urinary frequency and dysuria of approximately two weeks duration. Physical examination was essentially negative. Urine was cloudy-amber in appearance, acid in reaction, with a specific gravity of 1.016, plus 1 albumin, and 20-30 W. B. C.'s per hpf with numerous bacteria visible on microscopic examination. Urinary culture revealed a mucoid *Aerobacter aerogenes* and non-hemolytic *Streptococcus fecalis.* A urine sediment smear was loaded with gram-negative rods and a few gram-positive cocci in short chains. Patient was given 40,000 units of penicillin intramuscularly q. 4 h. for a total of 2.4 million units, a urinary antiseptic for 28 days, and sulfathiazole for 14 days, (total 21 gm.). Bladder irrigation was performed daily for 28 days with a solution of 1:8000 $KMnO_4$ and instillation of one ounce of a mild silver protein. Patient was discharged October 16, 1945, with a slight clinical improvement, but with an unchanged laboratory picture of the urinary infection.

On October 25, 1945, patient was re-admitted with repeated urinary symptomatology and arthritic-like pains in knee, ankle, elbow, and shoulder joints. Diathermy treatment applied to involve extremities; and the following therapy was carried out: Thiamine chloride 100 mg. daily, testosterone propionate 10 mg. a. o. d., and bladder irrigation q. o. d. with 1:8000 solution $KMnO_4$ followed by instillation of one ounce of a mild silver protein. An intravenous program during this admission showed normal kidneys. Urine was cloudy-yellow, alkaline, specific gravity of 1.015, albumin plus 1, and 10-15 W. B. C.'s per hpf. Patient was discharged on November 10, 1945, unimproved.

Patient re-admitted December 12, 1945, complaining of arthritic pains and urinary distress as on previous admission, and was given theelin capsules one q. i. d., a urinary antiseptic, testosterone propionate ampule one twice weekly, gold salt 5 mg. once weekly, and penicillin 40,000 units q. 4 h. for a total dosage of 1.8 million units. Patient was discharged on January 7, 1946, unimproved.

Patient was again re-admitted on January 27, 1946, with repeated urinary and arthritic complaints. Urinalysis and urine culture showed a continued picture of urinary infection with a mucoid *Aerobacter aerogenes.* All medication was withdrawn from patient and treatment began with streptomycin.

TREATMENT PLAN

7.0 grams* of stretpomycin sulfate was obtained**, 0.5 grams per ampule. At daily intervals, contents of one ampule was dissolved in 10 cc. of sterile physiological saline (equivalent to 50 milligrams per cc.) and 400 millgrams of this injected intramuscularly every 4 hours for the entire treatment period. Blood was obtained for streptomycin levels, and catherized urine was obtained for culture, at intervals indicated (Chart I). The invading organism was a mucoid *Aerobacter aerogenes,* which showed an *in vitro* sensitivity of 0.3 micrograms of streptomycin per cc.

* Pure crystalline streptomycin base, the activity of which was formerly designated in units, is now expressed in terms of weight. For practical purposes, 1 unit is the antibacterial activity of 1 microgram (0.001 milligrams), therefore 7 grams is the equivalent of 7 million micrograms, or 7 million units.

** Through the courtesy of Dr. H. Sidney Newcomer, E. R. Squibb & Sons, N. Y. C.

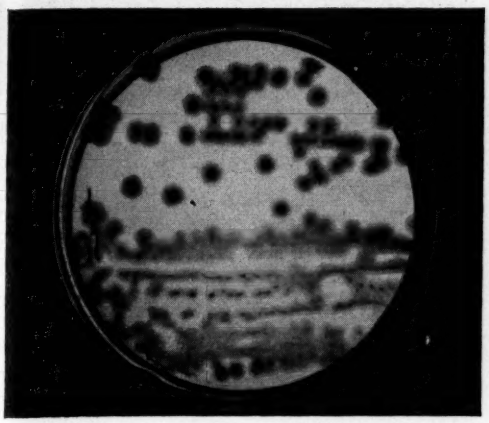


Fig. I—EMB plate streaked with urine sediment before treatment with Streptomycin. Note the large mucoid colonies of A. aerogenes. (½ actual size).

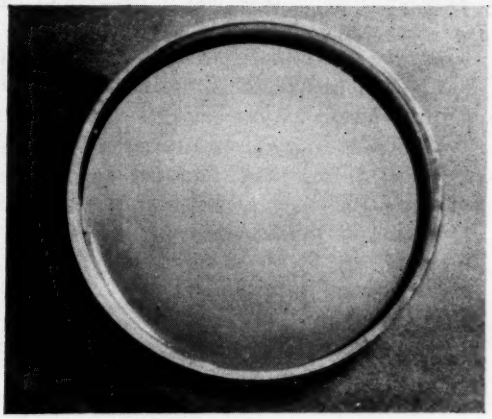


Fig. II—EMB plate streaked with urine sediment after treatment with Streptomycin. Note the absence of growth. (½ actual size).

INITIAL LABORATORY FINDINGS

Stained Urinary Sediment: Loaded with pus cells and gram-negative rods.

Culture. Mucoid Aerobacter aerogenes.

Complete Blood Count: RBC 4.4 million; Hemoglobin 13 grams, 83%; WBC 8,900; Differential-Polys 78%; Lymphocytes 20%; Monocytes 1%; Eosinophiles 1%.

Blood Urea Nitrogen: 15.8 mg.%.

Total Serum Protein: 5.1 gm.%.

Sedimentation Rate: 22 mm. in 1 hour.

CHART I

LABORATORY FINDINGS DURING TREATMENT

Date	Drug	Total Given (grams)	1 Hour Serum Level* (Micrograms/cc)	Laboratory Tests
1/28		0.4	40	
1/29 A.M.		2.4	50	Stained Urinary Sediment: Negative. Culture: Negative after 48 hours.
1/29 P.M.			55	Blood Urea Nitrogen: 13.8mg.% Plasma Chlorides: 572 mg.%
1/30 A.M.		4.4	55	Stained Urinary Sediment: Negative. Culture: Negative after 48 hours.
1/30 P.M.			58	
1/31 A.M.		6.8	55	Stained Urinary Sediment: Negative. Culture: Negative after 48 hours.
2 /1 A.M.			25	
2 /2			0	Sugar: 107 mg.% Blood Urea Nitrogen: 18.5 mg.%
2 /6			0	Stained Urinary Sediment: Negative. Culture: Negative after 48 hours.

* Performed by R. H. Fowler, Bacteriologist, Abington Memorial Hospital, Pa., using Stebbins and Robinson technic 4.

DISCUSSION

In brief, the foregoing material presents the picture of a 77-year-old, white male patient suffering with arthritic pains and a urinary frequency and dysuria due to urinary tract infection by *Aerobacter aerogenes.*

Over a period of six months the patient was treated with courses of penicillin and sulfathiazole therapy; a urinary antiseptic; bladder irrigation with $KMnO_4$ and mild silver protein instillation; diathermy; thiamine chloride; testosterone propionate; and gold salt medications. All of this therapy was of little or no avail in alleviating the patient's symptoms or improving the laboratory picture of the infectious process.

A course of treatment of 6.8 grams of streptomycin sulfate was given in divided dosage, over a period of 72 hours. Before completion of the instituted therapy, a sterile urine culture was obtained, together with mitigation of the clinical urinary complaints. The final result was a patient free of urinary tract symptomatology, and an amelioration of his arthritic pains.

SUMMARY

1. A case of long-standing urinary tract infection due to *Aerobacter aerogenes* is presented.

2. The organism was resistant, *in vivo,* to penicillin, sulfathiazole, and common urinary antiseptics, but sensitive, *in vitro,* to 0.3 micrograms of streptomycin per cc.

3. A prompt bacteriological cure was effected by the parenteral administration of 6.8 grams of streptomycin sulfate, given over a 72 hour period. Substantial blood levels were attained.

4. Apart from the local discomfort of intramuscular injection, there was no serious or uncontrollable toxic effect from the use of streptomycin in this case.

REFERENCES

1. Herrell, W. E., and Nichols, D. R.: Proc. Staff Meet., Mayo Clinic, 20:449, 1945.
2. Reimann, H. A., Price, A. H., and Elias, W. F.: Arch. Int. Med., 76:269, 1945.
3. Foshay, L., and Pasterneck, A. B.: J. A. M. A., 130: 393, 1946.
4. Stebbins, R. B., and Robinson, H. J.: Proc. Soc. Exp. Biol. & Med., 59:255, 1945.

ROCKY MOUNTAIN SPOTTED FEVER: A Case Treated With p-Aminobenzoic Acid*

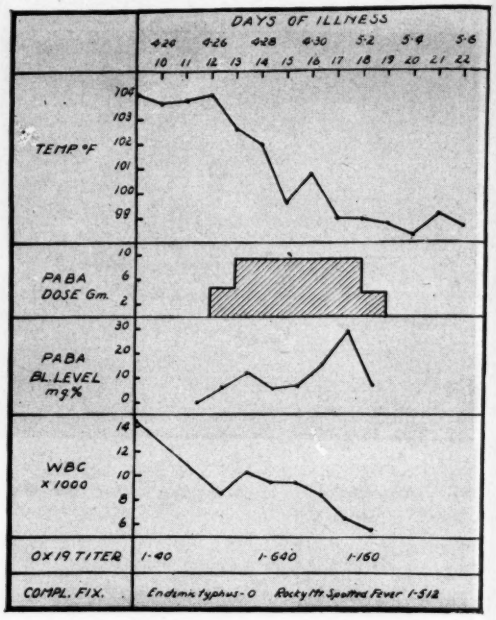
J. W. MARONEY, M. D., H. C. DAVIS, M. D., AND E. G. SCOTT, M. T., Wilmington, Del.

Rose and his co-workers[1] were the first to report a human case of Rocky Mountain spotted fever treated with benefit by p-aminobenzoic acid. This clinical report followed the experimental work of Anigstein and Bader[2] and Hamilton[3] who reported that the drug proved therapeutically effective in guinea pigs infected with Rocky Mountain spotted fever rickettsias. The drug inhibited these rickettsias in chick embryos to an even greater extent than typhus rickettsias.

Recently it was our opportunity to observe a clincal case of this disease, and treat it with p-aminobenzoic acid. Administration of the drug 12 days after onset was followed by a prompt defervescence of the temperature, signs and symptoms, and no toxic manifestations were observed. This case is therefore presented as an incentive to further studies of the use of p-aminobenzoic acid in this disease.

CASE REPORT

S. H., a three-year-old colored female, was admitted to the pediatric service on April 23, 1946. Eight days prior to admission she had suddenly developed a fever of 104 F. A diagnosis of grippe was made, and a sulfonamide was administered without effect on the fever. Three days later a rash was noted on



Course of Disease.

the thighs, hands and wrists, spreading to the trunk. The face was not involved. The rash was first considered to be a reaction to the sulfonamide. The child showed weight loss, constipation, anorexia, irritability and prostration. No history of tick bite could be elicited. The child, however, lived in a tick-infested area, and spent most of her time playing out of doors, sometimes walking in the tall grass of a near-by playground.

On admission, the patient was irritable, and cried easily when disturbed. The temperature was 104 F. by rectum, the pulse 160, the respirations 32, and the blood pressure was 109 systolic and 74 diastolic.

The skin was hot and dry, with a purpuric rash over the extremities and trunk. The individual lesions were discrete, averaging 1-3 mm. in diameter, purplish-red in color, appeared to be deep rather than superficial, and blanched readily on pressure. The face was essentially uninvolved. There was generalized lymphadenopathy. The lips were bleeding slightly, the buccal mucosa was clear. The heart and lungs were normal, the liver slightly enlarged to percussion. While the rash most closely resembled a rickettsial infection, both

* From the Departments of Pediatrics and Pathology, Delaware Hospital.

a toxic purpura and a meningococcemia were considered in the differential diagnosis.

On admission the blood count showed 3.8 million RBC, 10 gm. hemoglobin 14,400 WBC, 75% polys, and 244,000 platelets. Urinalysis showed a 1 plus albumin and 8-15 WBC/HPF. A lumbar puncture showed a clear colorless fluid under increased pressure, with normal protein and chlorides, and 28 cells per cu. mm. (predominately mononuclears). No organisms were seen in the stained sediment, and cultures remained sterile for 72 hours. No growth occurred on aerobic or anaerobic blood cultures after 10 days incubation. Febrile agglutinins were negative for typhoid, paratyphoid and brucella antigens, and positive with proteus OX19 antigen thru a titer of 1:40. A clinical diagnosis of Rocky Mountain spotted fever was then made. For further confirmation, the patient's serum was submitted to Dr. Charles Armstrong, Chief, Division of Infectious Diseases, National Institute, Bethesda, Md., on April 28th (14th day of illness) for rickettsial studies. Dr. Armstrong reported that complement fixation tests with endemic typhus antigens were negative, but showed a positive titer of 1:512 or higher, with Rocky Mountain spotted fever antigen. He reported further that the patient's serum agglutinated proteus OX19 antigen thru a titer of 1:640. A repeat OX19 agglutination was performed by us on May 1st (17th day of illness) and the titer had risen from 1:40 to 1:160.

Therapy with p-aminobenzoic acid was started three days after admission (12th day of illness). The method of administration is reported in detail. The drug was prepared in packets containing an initial dose of 1.3 grams, and subsequent doses of 0.8 grams each. These, along with a large bottle of 5% sodium bicarbonate, were sent to the ward with instructions to dissolve each packet in one-half ounce of chilled bicarbonate solution, this dose to be given orally every two hours. The dosage was intended to approximate one-third of that used by Yeomans[4] and Rose in an adult, in an attempt to maintain a blood level of 10-20 mg.%. P-aminobenzoic acid is relatively insoluble in water and quite soluble in 5% sodium bicarbonate, but considerable difficulty was encountered in administering it to a three-year-old patient. At the suggestion of Dr. C. W. Todd, Chemical Consultant to the Department of Pathology, 0.8 grams was found to be somewhat soluble in one ounce of orange juice at room temperature, provided it was prepared about two hours before administration. This was given with some degree of success, but Levine tube administration was finally resorted to. After several such feedings the patient took the oral dose without further difficulty, probably coincident with decreased irritability and general improvement in her condition.

Daily p-aminobenzoic acid blood levels, leukocyte counts, hemoglobin estimations, and urinalyses (when obtainable) were performed while the drug was being administered. The technic for p-aminobenzoic acid was modified by Dr. Todd from the Bratton & Marshall method for sulfonamide estimation, using 0.2 ml. of blood obtained by finger puncture. These levels ranged from 5.1 to 29.1 mg.%. The white count fell from 14,400 to 8,500 in 72 hours after medication was initiated, and remained within the normal range thereafter. Urine specific gravities varied from 1.012 to 1.019, and a 1 plus albumin persisted during treatment, with a normal urinalysis at discharge. Hemoglobin values ranged from 10 to 8.1 gm.

On the second day of p-aminobenzoic acid therapy, the temperature, which had ranged from 102 to 104 F., fell to normal, with some fading of the rash. On the third day the rash had faded markedly, changing from purplish-red to sandy brown. The drug was continued for eight days without any apparent toxic manifestations. Clinical improvement accompanied the falling temperature, and the childs' appetite and good spirits were rapidly regained. The patient was discharged in good health on the fourteenth hospital day.

COMMENT

The recovery of this child with clinically typical and serologically demonstrated Rocky Mountain spotted fever following administration of p-aminobenzoic acid was most dramatic. In an area where this disease presents a constant menace during the tick season, we have not previously seen so abrupt a defervescence of symptoms. While we cannot

draw conclusions from an isolated case, it was our clinical impression that recovery was enhanced by the drug, and that it merits further trial.

SUMMARY

A case of Rocky Mountain spotted fever treated with p-aminobenzoic acid is reported, with an apparent beneficial effect.

REFERENCES

1. Rose, H. M., Duane, R.B., and Fischel, E. E.: J. A. M. A., 129:1160, 1945.
2. Anigstein, L., and Bader, M. N.: Science, 101:591, 1945.
3. Hamilton, H. L.: Proc. Soc. Exper. Biol. & Med., 59:220, 1945.
4. Yeomans, A., Snyder, J. C., Murray, E. S., Zarafonetis, C. D. J., and Ecke, R. S.: J. A. M. A., 126: 349, 1944.

NEW LIGHT ON FILTERABLE VIRUS

The filterable virus, probably mans' most deadly enemy, is a highly complex structure.

New light on the nature of the almost infinitesimally minute things which are responsible for some of the most dreaded human and animal diseases has been obtained from studies at Duke University, according to a report just made to the Office of the Surgeon General of the Army under whose direction experimental work was conducted during the war.

The viruses have diameters of only a few millionths of a millimeter. They are far below the limits of the most powerful optical microscope. Through use of the electron microscope and microchemical techniques, however, it was possible for the Duke investigators to obtain considerable information.

They are so minute that there has been some question as to whether they are actual living things, or large molecules somehow endowed with the ability to reproduce themselves.

But, says Dr. Joseph W. Beard who was in charge of the Duke investigations under the Army: "These particles cannot be molecules. They are of very complex structure and apparently are enclosed in a membrane."

The studies were made on two viruses—one of which causes a disease of rabbits known as papilloma and the other the human malady vaccinia—and one of the bacteriophages, which are quite similar organizations. These were simpler to study than the influenza viruses which were the ultimate objectives of the Duke investigations. It was felt that any knowledge of viruses in general ultimately might prove of value.

The bacteriophage especially looked like an ultramicroscopic tadpole. It has a well-defined head and a stubby tail. The papilloma virus was spheroidal in shape while the vaccinia organism was like a flattened disc with denser internal material bulging beneath the surface of its "skin."

Other tests showed that these viruses were a little more than half water. The chemical composition of the bacteriophage consisted of a mixture of proteins and lipoids, or basic constituents of fats, in association with a high content of nucleic acids, very complex compounds found in the nucleii of all living cells. The chief element was carbon—about 42 per cent. There also were considerable amounts of nitrogen and phosphorus. The diameter of the papilloma virus was found to be about 45 thousandths of a millimeter.

The work has just been reported through the Army Epidemiological Board.

We give our medical students innumerable facts from which they should infer that the periodic health examination is important. Yet, partly because we do not carry this education through to its behavior aspects as related to them, physicians themselves seldom have such examinations. They are more likely than most people to develop active tuberculosis, and have little advantage over others in recognizing an early case in themselves. Yet how few have periodic X-rays. Those who smile self-righteously at the physicians might reflect, however, that the failing is common elsewhere. Some time ago the Minnesota Supreme Court, in handing down an opinion, remarked that lawyers, although recognizing the dangers common when no will has been made, notoriously seldom have made wills themselves.—Carl J. Potthoff, M. D., *Amer. Jour. P. H.*, Oct., 1945.

1789—MEDICAL SOCIETY OF DELAWARE—1946

OFFICERS

PRESIDENT, Walter C. Deakyne, Smyrna
FIRST VICE-PRESIDENT, Howard S. Riggin, Seaford
SECOND VICE-PRESIDENT, W. Edwin Bird, Wilmington
SECRETARY, C. Leith Munson, Wilmington
TREASURER, William W. Lattomus, Wilmington

COUNCILORS

Joseph B. Waples, Georgetown (1946) Jerome D. Niles, Townsend (1947) Clarence J. Prickett, Smyrna (1948)

AMERICAN MEDICAL ASSOCIATION

DELEGATE: James Beebe, Lewes (1947) ALTERNATE: Clyde C. Neese, Wilmington (1947)

STANDING COMMITTEES

SCIENTIFIC WORK
H. V'P. Wilson, Dover
E. L. Stambaugh, Lewes
C. L. Munson, Wilmington

PUBLIC POLICY AND LEGISLATION
J. S. McDaniel, Dover
James Beebe, Lewes
J. D. Niles, Townsend

PUBLICATION
W. E. Bird, Wilmington
M. A. Tarumianz, Farnhurst
C. L. Munson, Wilmington

MEDICAL EDUCATION
L. B. Flinn, Wilmington
K. S. Brickley, Lewes
I. J. MacCollum, Wyoming

NECROLOGY
E. M. Vaughan, Middletown
Wm. Marshall, Jr., Milford
U. W. Hocker, Lewes

ADVISORY COMMITTEE WOMEN'S AUXILIARY
H. N. Stayton, Rehoboth
Catherine Gray, Bridgeville
C. B. Scull, Dover
W. T. Chipman, Harrington
A. L. Heck, Wilmington

REPRESENTATIVE TO THE DELAWARE ACADEMY OF MEDICINE
W. O. LaMotte, Wilmington

CANCER
J. F. Hynes, Wilmington
J. W. Howard, Wilmington
D. M. Gay, Wilmington
W. H. Kraemer, Wilmington
H. V'P. Wilson, Dover
O. A. James, Milford
James Beebe, Lewes
C. J. Prickett, Smyrna
J. R. Elliott, Laurel

SYPHILIS
B. S. Vallett, Wilmington
Bruce Barnes, Seaford
Stanley Worden, Dover

TUBERCULOSIS
L. D. Phillips, Marshallton
L. B. Flinn, Wilmington
G. A. Beatty, Wilmington
G. W. K. Forrest, Wilmington
L. J. Jones, Wilmington
C. C. Fooks, Milford
C. M. Moyer, Laurel
L. L. Fitchett, Milford
R. C. Comegys, Clayton

MATERNAL AND INFANT MORTALITY
E. L. Stambaugh, Lewes
C. L. Hudiburg, Wilmington
W. C. Pritchard, Jr., Smyrna
Stites McDaniel, Dover

MENTAL HEALTH
K. M. Corrin, Wilmington
I. J. MacCollum, Wyoming
James Beebe, Lewes

CRIMINOLOGIC INSTITUTES
H. M. Manning, Seaford
M. A. Tarumianz, Farnhurst
Wm. Marshall, Jr., Milford

SPECIAL COMMITTEES

REVISION OF BY-LAWS
W. E. Bird, Wilmington
E. R. Miller, Wilmington
C. E. Wagner, Wilmington
J. S. McDaniel, Dover
R. C. Beebe, Lewes

MEDICAL ECONOMICS AND PUBLIC RELATIONS
J. S. McDaniel, Dover
W. E. Bird, Wilmington
W. H. Speer, Wilmington
E. R. Mayerberg, Wilmington
J. D. Niles, Middletown
L. B. Flinn, Wilmington
W. T. Chipman, Harrington
J. B. Waples, Georgetown
James Beebe, Lewes

VOCATIONAL REHABILITATION
W. H. Speer, Wilmington
H. V'P. Wilson, Dover
Wm. Marshall, Milford
U. W. Hocker, Lewes
F. I. Hudson, Rehoboth

POST-WAR PLANS
M. A. Tarumianz, Farnhurst
W. E. Bird, Wilmington
W. H. Speer, Wilmington
W. O. LaMotte, Wilmington
J. S. McDaniel, Dover
Wm. Marshall, Jr., Milford
J. R. Elliott, Laurel
J. B. Waples, Georgetown
R. C. Beebe, Lewes

BUDGET
M. A. Tarumianz, Farnhurst
C. E. Wagner, Wilmington
J. D. Niles, Townsend
J. S. McDaniel, Dover
James Beebe, Lewes

CHEFF MEMORIAL
W. W. Lattomus, Wilmington
E. R. Miller, Wilmington
A. J. Heather, Wilmington

WOMAN'S AUXILIARY

MRS. E. L. STAMBAUGH, *President*, Lewes
MRS. G. C. MCELFATRICK, *Vice-Pres. for N. C. County*, Wilmington
MRS. I. J. MACCOLLUM, *Vice-Pres. for Kent County*, Wyoming
MRS. JAMES BEEBE, *Vice-Pres. for Sussex County*, Lewes
MRS. S. W. RENNIE, *Recording Secretary*, Wilmington
MRS. K. S. BRICKLEY, *Corresponding Secretary*, Lewes
MRS. A. J. STRIKOL, *Treasurer*, Wilmington

NEW CASTLE COUNTY MEDICAL SOCIETY—1946

Meets Third Tuesday

IRA BURNS, *President*, Wilmington.
ROGER MURRAY, *President-elect*, Wilmington.
R. O. Y. WARREN, *Vice-President*, Wilmington.
E. R. MILLER, *Secretary*, Wilmington.
CHARLES LEVY, *Treasurer*, Wilmington.

Board of Directors and Nominating Committee: A. J. Strikol, 1946; C. C. Neese, 1947; L. B. Flinn, 1948.

Delegates: 1946: W. E. Bird, Ira Burns, G. H. Gehrmann, J. F. Hynes, L. J. Jones, E. R. Mayerberg, E. R. Miller, C. C. Neese, A. J. Strikol, M. A. Tarumianz. 1946-47: B. M. Allen, L. W. Anderson, G. A. Beatty, L. B. Flinn, G. W. K. Forrest, J. D. Niles, S. W. Rennie, F. S. Skura, M. W. Squires, C. E. Wagner.

Alternates: 1946: J. W. Butler, W. W. Ellis, Mildred Forman, Margaret Handy, A. L. Heck, T. V. Hynes, E. M. Krieger, J. M. Messick, J. C. Pierson, P. R. Smith. 1946-47: D. D. Burch, I. L. Flinn, Catherine Johnson, E. C. Laird, C. E. Maroney, L. C. McGee, W. F. Preston, W. T. Reardon, W. J. Urie.

Board of Censors: A. L. Heck, 1946; B. M. Allen, 1947; N. W. Voss, 1948; C. L. Hudiburg, 1949; C. L. Munson, 1950.

Program Committee: Roger Murray, Ira Burns, R. O. Y. Warren.

Legislative Committee: W. O. LaMotte, M. A. Tarumianz, J. M. Barsky, T. H. Baker, C. M. Bantroft.

Public Relations Committee: C. E. Wagner, B. A. Gross, G. W. K. Forrest, M. B. Pennington, R. A. Lynch.

Medical Economics Committee: W. E. Bird, J. R. Durham, Jr., Verna S. Young, W. M. Pierson, L. W. Anderson.

Necrology Committee: K. M. Corrin, P. A. Shaw, W. H. Lee.

Auditing Committee: A. J. Heather, V. C. Nah, A. R. Cruchley.

KENT COUNTY MEDICAL SOCIETY—1946

Meets Second Wednesday

FRANKLIN R. EVERETT, *President*, Dover.
JOHN P. MARTIN, *Vice President*, Camden.
EUGENE H. MERCER, *Secretary-Treasurer*, Dover.

Delegates: *Jos. McDaniel*, Dover; H. W. Smith, Harrington.
Alternates: I. J. MacCollum, Wyoming; John Martin, Camden.
Censor: W. C. Deakyne, Smyrna.

DELAWARE ACADEMY OF MEDICINE—1946

Open 10 A. M. to 1 P. M.

EDGAR R. MILLER, *President*.
GERALD A. BEATTY, *First Vice-President*.
DOUGLAS T. DAVIDSON, SR., *Second Vice-President*.
ROBERT R. WIER, *Secretary*.
IRVIN M. FLINN, *Treasurer*.
Board of Directors: S. D. Townsend, L. B. Flinn M. D.; C. M. A. Stine, J. K. Garrigues, W. S. Carpenter, Jr., H. A. Carpenter, Mrs. Ernest du Pont, W. H. Kraemer, M. D.

DELAWARE PHARMACEUTICAL SOCIETY—1946

F. PERRY RAGAINS, *President*, Milford.
CHARLES HOPKINS, *First Vice-President*, Dover.
THOMAS DAVIS, *Second Vice-President*, Wilmington.
G. J. MCNAUGHTON, *Third Vice-President*, Middletown.
ALBERT BUNIN, *Secretary*, Wilmington.
ALBERT DOUGHERTY, *Treasurer*, Wilmington.
Board of Directors: F. Perry Ragains, C. Emerson Johnson, Al. S. Williams, Everett D. Bryan, Vernon L. Larson.
Chairman, Legislative Committee: Thomas Donaldson.

MEDICAL COUNCIL OF DELAWARE

Hon. Charles S. Richards, *President*; **Joseph S. McDaniel, M. D., *Secretary*;** Wallace M. Johnson.

SUSSEX COUNTY MEDICAL SOCIETY—1946

Meets Second Tuesday—Even Months

A. H. WILLIAMS, *President*, Laurel.
V. A. HUDSON, *Vice-Pres.*, Millsboro.
R. S. LONG, *Secretary-Treasurer*, Frankford.

Delegates: Bruce Barnes, R. S. Long, James Beebe, H. S. Riggin.

Alternates: L. M. Dobson, C. M. Moyer, E. L. Stambaugh, G. W. M. Van Valkenburgh.

Censors: O. A. James, R. C. Beebe, A. C. Smoot.

DELAWARE STATE DENTAL SOCIETY—1946

BLAINE ATKINS, *President*, Millsboro.
FRANK M. HOOPES, *First Vice-President*, Wilmington.
HENRY S. KEAVENY, *Second Vice-President*, Wilmington.
JAMES KRYGIER, *Secretary*, Dover.
LOUIS GLUCKMAN, *Treasurer*, Wilmington.
PHILIP A. TRAYNOR, *Del., A. D. A.* Wilmington.

DELAWARE STATE BOARD OF HEALTH—1946

J. D. Niles, M. D., *President*, Middletown; Mrs. F. G. Tallman, *Vice-Pres.*, Wilmington; W. B. Atkins, D. D. S., *Secretary*, Millsboro; Bruce Barnes, M. D., Seaford; Mrs. C. M. Dillon, Wilmington; W. T. Chipman, M. D., Harrington; Mrs. Alden Keene, Middletown; E. R. Mayerberg, M. D., Wilmington; Edwin Cameron, M. D., *Executive Secretary*, Dover.

BOARD OF EXAMINERS, MEDICAL SOCIETY OF DELAWARE

J. S. McDaniel, *President and Secretary*; Wm. Marshall, *Assistant Secretary*; W. E. Bird, W. T. Chipman, P. R. Smith.

A NEW CEREAL FOR

Infants and Children WITH PAPAYA FRUIT

DRIED FRESH TO PRESERVE NATURAL ENZYMES AND PECTINS

Cerol

THE LATEST ADDITION to the famous S. M. A. Infant Foods —CEROL . . . something new in infant feeding—FLAVORED . . . with mellow papaya fruit—FORTIFIED . . . with vitamins and minerals — READY TO SERVE . . . a nutritious, precooked, multigrain cereal—Supplied in 8 oz. packages.

Wyeth

REG. U. S. PAT. OFF.

S. M. A. DIVISION • WYETH INCORPORATED • PHILADELPHIA 3 • PA.

+ Editorial +

DELAWARE STATE MEDICAL JOURNAL

Owned and published by the Medical Society of Delaware, a scientific society, non-profit corporation. Issued about the twentieth of each month under the supervision of the Committee on Publication.

W. EDWIN BIRD, M. D. ..... Editor
822 North American Building

W. OSCAR LA MOTTE, M. D. ..... Associate Editor
Medical Arts Building

M. A. TARUMIANZ, M. D. .....Assoc. & Managing Editor
Farnhurst, Del.

Articles are accepted for publication on condition that they are contributed solely to this JOURNAL. Manuscripts must be typewritten, double spaced, with wide margins, and the original copy submitted. Photographs and drawing for illustrations must be carefully marked and show clearly what is intended.

Footnotes and bibliographies should conform to the style of the Quarterly Cumulative Index Medicus, published by the American Medical Association, Chicago.

Changes in manuscript after an article has been set in type will be charged to the author. THE JOURNAL pays only part of the cost of tables and illustrations. Unused manuscripts will not be returned unless return postage is forwarded. Reprints may be obtained at cost, provided request is made of the printers before publication.

The right is reserved to reject material submitted for publication. THE JOURNAL is not responsible for views expressed in any article signed by the author.

All advertisements are received subject to the approval of the Council on Pharmacy and Chemistry of the A. M. A. Advertising forms close the 25th of the preceding month.

Matter appearing in THE JOURNAL is covered by copyright. As a rule, no objection will be made to its reproduction in reputable medical journals, if proper credit is given. The reproduction in whole or in part, for commercial purposes of articles appearing in THE JOURNAL will not be permitted.

Subscription price: $2.00 per annum, in advance. Single copies, 20 cents. Foreign countries: $2.50 per annum.

VOL. 18 MAY, 1946 No. 5

RE: BY-LAWS

Revision of the By-Laws of the Medical Society of Delaware, which was initiated by the House of Delegates in 1941, was later held in abeyance by order of the House, till the large percentage of our membership who went into the services returned. That glad day is about here, there being only a comparatively small number of our men yet to return. Hence the time to begin work on this much-needed revision has also nearly arrived. We have the assurance of the Bureau of Legal Medicine of the American Medical Association that whenever we are ready to proceed they will send a representative here to advise us. This asistance will be much appreciated, for, with certain peculiar ideas prevailing in the national law-making bodies, there are hidden dangers in writing even such an apparently innocent item as the Preamble, especially in the definition of the purposes of the Society.

It is the hope of the Committee on Revision of the By-Laws to have this matter completed in time to present to the House of Delegates next October.

Likewise, certain changes in the By-Laws of the New Castle County Medical Society have been proposed, and others will be necessitated by the changes in the State By-Laws. When these latter have been made, it is hoped to complete the County By-Laws before the end of the year. It is almost certain that the Kent County and Sussex County Societies will also find the necessity of adjusting their present By-Laws to the new State ones. Thus, by the end of this year the State Society and its component county societies will have revised and modernized their By-Laws, a long overdue task.

HEALTH AND ACCIDENT INSURANCE

Recently the Kent and Sussex County Medical Societies have jointly contracted with a commercial carrier for a group policy for health and accident coverage. There are certain advantages in a group contract not available to the individual subscriber, and a sufficiently large percentage of the membership of the Kent and Sussex Societies subscribed to justify a group contract.

In the New Castle County Medical Society the Committee on Medical Economics has presented this subject periodically for the past five or six years. However, due to the war and to the fact that a fair percentage of the membership already had this type of coverage, no interest was manifested in a group contract. It was presented again at the April meeting, and the same lack of interest was exhibited. We do believe, though, that the members of this Society should give the matter their serious thought during the summer; in the early fall it will again .be presented for their decision.

SOCIALIZED MEDICINE ABROAD

The proponents of socialized medicine, often enthusiasts or even fanatics on the subject, usually try to sugar-coat Government medicine in order to gain converts. They even try to make it attractive to physicians. Sometimes they propose laws under which medical practitioners would receive remuneration better than the average under today's system of free enterprise. Of course, these proponents know that once they get their bills for socialized medicine enacted into law not even dynamite could remove the incubus. (Remember how long it took to get rid of National prohibition and how for so long it seemed impossible to get rid of it.) Once socialized medicine got on the statute books no one would need to be appeased to keep it there; certainly not the physicians who are too small a minority group among the voters to make their opinions felt politically; neither would the politicians need to appease the vast public who are the patients and who would think they were getting medical service for nothing even while paying exorbitant prices for inferior goods.

Great Britain has had socialized medicine for many years. The panel medicine the British public has been receiving has done not one of the good things, such as adequate medical service, which is claimed for socialized medicine but, nevertheless, the failure of this type of medical practice to be anywhere near the equal of untrammeled free enterprise is not bringing about the withdrawal of the Government from medical practice. On the contrary, Britain's Labor Government now demands even greater socialization of medical practice. (One wonders if the proposed loan by the U. S. to Great Britain is to help defray its cost.) The Scottish Daily Express, founded by Lord Beaverbrook, under date of March 22, 1946, has the following news item on the subject, which is quoted in part:

Britain's 56,000 doctors will receive today from the British Medical Association a call to fight Mr. Bevan's plan to nationalize doctors.

B. M. A. officials worked late last night to send the call by post to every doctor, with a copy of the National health service White Paper.

The "Come Out and Fight" campaign will be Nation-wide. Meetings are being arranged all over the country during the first fortnight of next month to challenge—

Conversion of doctors into civil servants.

The new health centres shared by six to ten doctors.

The administrative "badness" of the Bill.

The first big meeting of doctors will be held at Wimbledon Town Hall, London, next Sunday, when Dr. Charles Hill of the B. M. A., will speak to about 200 doctors.

The Bill leaves seven Government departments all operating medical services.

The collapse of the old doctor-patient relationship horrifies many of the doctors. They will warn the people that if the Bill goes through the doctor must work under official approval.

"In the campaign, the public will be told that the Bill means an era of divided loyalties—the patient or the State? The doctor, largely paid by State salary, will bring trouble his way if he fights a patient's claim against the Government."

The B. M. A. hopes to mobilize public opinion against the State control of the hospitals. Its members believe in the local tradition enjoyed by many voluntary hospitals. They consider there is a danger that small but cherished hospitals may be closed in the name of administrative thoroughness.

The B. M. A. is angry over the way Mr. Bevan has dealt with it, and complains that previous Ministers of Health asked the B. M. A. Council for its views on health service questions.

Dr. H. Guy Dain, chairman of the council, said last night: "When Mr. Bevan did see us, he told us—'I shall be grateful for your comments, but I am not proposing to negotiate with you, but I shall take entire responsibility for the form of the Bill.'

"We offered comments, but we have not yet discovered what consideration he gave them or whether they affected his views."

Dr. Charles Hill, B. M. A. secretary, said: "The White Paper suggests that the doctor-patient relationship will not be impaired. That is not accurate. The proposals lead sooner, rather than later, to the doctor becoming a whole-time salaried civil servant.

"We particularly object that a doctor should be directed to a particular area."

Thus it goes. "Let's have socialized medicine," says it proponents, "no matter how far we must go in making concessions to get it. Once we have it, we can mold it to suit our purposes and need pay no attention to objectors." The time to stop socialized medicine is now before it becomes law. If we wait too long we'll probably live with it for the rest of our lives.—Editorial, *Phila. Med.*, May 11, 1946.

BOOK REVIEWS

Gastro-Enterology. By Henry L. Bockus, M. D., Professor of Gastro-Enterology, University of Pennsylvania Graduate School of Medicine. In three volumes, totaling about 2700 pages with about 900 illustrations, many in colors. Volume III—The Liver, Biliary Tract and Pancreas, and Secondary Gastro-Intestinal Disorders. Pp. 1091, with 427 illustrations, some in colors. Cloth. Price (3 vols. and separate desk index) $35.00. Philadelphia: W. B. Saunders Company, 1946.

Volume I of this splendid work was reviewed in THE JOURNAL of September, 1943, and Volume II in March, 1944. Now appears the final volume, with separate check index. This one contains 29 chapters contributed by 14 of Dr. Bockus' associates, and 8 others written jointly, leaving 11 chapters exclusively by Bockus. The remarkable thing is the continuity of style—we could not tell whose chapter we were reading.

It would be difficult to point to any chapter that stands out alone, such is the uniform excellence of the whole. While this is primarily a medical text, its surgical advice, even including some technique, represents the best of the surgical thinking of today. These combined features make this work, in our opinion, the outstanding one in its field—we know of nothing in the English language that equals it.

The Management of Fractures, Dislocations and Sprains. By John Albert Key, M. D., Clinical Professor of Orthopedic Surgery, Washington University, and H. Earle Conwell, M. D., Orthopedic Surgeon, Tennessee Coal, Iron and Railroad Company. Fourth Edition. Pp. 1322, with 1318 illustrations. Cloth. Price, $12.50. St. Louis: C. V. Mosby Company, 1946.

When a book is so popular as to require a new edition every four years, it has to be good. Key and Conwell have produced a work that, to hundreds of American physicians, is a veritable Bible within its field. This new edition brings the subject completely up-to-date, including the use of penicillin and other chemical and biological agents. Important changes have been made in the sections on the spine, the hip and compound fractures. To us, the most interesting change is in the section on intervertebral disc, now expanded to eight pages, and one of the best discussions of this sometimes baffling subject, in which the two authors very politely disagree with each other. The authors describe at length the treatments that in their hands have produced the most satisfactory results, which is to say that the text is both practical and dependable.

The volume is too encyclopedic to review in detail, but our old regard for it still persists—text plus illustrations make it one of the really outstanding books in its field.

Preoperative and Postoperative Treatment. Edited by Lt. Col. Robert L. Mason, M. C., A. U. S., Cushing General Hospital, Framingham, Massachusetts; and Harold A. Zintel, M. D., Harrison Department of Surgical Research, University of Pennsylvania School of Medicine; Assistant Surgeon, Hospital of the University of Pennsylvania. Second Edition. Pp. 584, with 157 illustrations. Cloth. Price, $7.00. Philadelphia: W. B. Saunders Company, 1946.

Mason's book, which first appeared in 1937, has been practically rewritten. In addition, new chapters on physical medicine, care of gynecologic, gastric, duodenal and obstructed cases, nutrition, pregnancy, thrombophlebitis, vitamin K and cranial injuries appear. The editors secured 18 other writers to contri-

bute chapters within their special fields of interest, all of which are meritorious. The treatments described are in consonance with the latest concepts of modern physiology, biochemistry, bacteriology and pharmacology.

The book can be recommended without reserve.

Synopsis of Physiology. By Rolland J. Main, Ph. D., Professor of Physiology, Medical College of Virginia. Pp. 341, with 21 illustrations. Cloth. Price, $3.50. St. Louis: C. V. Mosby Company, 1946.

In response to the demand of physicians and senior medical students for "a little book which hits the high spots," Main has hit the main facts of physiology in this little book. Human, rather than animal, data are used wherever possible, and the normal is accentuated by brief references to the abnormal. In order to be as brief as possible, the author claims he has been shockingly dogmatic; if he has been, we seem to have missed it. The style is concise yet readable, and the arrangement convenient. We like this book very much.

Cornell Conferences on Therapy. Edited by Harry Gold, M. D., Assistant Professor of Pharmacology, Cornell University. Pp. 322. Cloth. Price, $3.25. New York: Macmillan Company, 1946.

These conferences were inaugurated in 1937 in an effort to bring pharmacologists and clinicians together, for their mutual understanding and the benefit of the patient. The scope covers the whole range of therapeutics; the aim has been to stimulate interest in rational therapy. They were published in the *Journal of the A. M. A.* from 1937 to 1940, and since that time in the *New York State Journal of Medicine*. The present volume, the first of an annual series, contains 15 conferences selected for their quality and lasting value, among them being: Use and Abuse of Bed Rest; Surgical Measures for the Relief of Pain; Treatment of Heart Failure; Treatment of Subacute Bacterial Endocarditis; Treatment of Poisoning; and the Rh-Factor in Therapy.

This volume, which will interest physicians, even surgeons, in all branches of therapy, is a valuable one. We look forward with anticipation for its successors.

A Bibliography of Infantile Paralysis: 1789-1944. Edited by Morris Fishbein, M. D., Editor, Journal of the American Medical Association. Pp. 672. Cloth. Price, $——, Philadelphia: J. B. Lippincott Company, 1946.

We were intrigued to note that "polio" was first described by Underwood in 1789, the very year that the Medical Society of Delaware, founded in 1776, was incorporated. We were not surprised to note that this bibliography is dedicated to the late Franklin D. Roosevelt. The one thing, perhaps the only thing, he personally licked was "polio," which was a notable victory for him at the time, and later on for all the polio victims, for with his exceptional "gift of gab" he popularized the nation-wide campaign against polio.

The book is excellent, is complete, except for the references to foreign literature during the war years, and includes abstracts of the more important articles. It will be of interest to research writers, to those who are concerned with the literature of polio, and to those who think that the late Roosevelt was the world's greatest symbol of battle against one of the world's greatest scourges.

Synopsis of Pathology. By W. A. D. Anderson, M. D., Professor of Pathology and Bacteriology, Marquette University School of Medicine. Second Edition, revised. Pp. 741, with 237 illustrations and 15 color plates. Cloth. Price, $6.50. St. Louis: C. V. Mosby Company, 1946.

This book is just what it claims to be, a synopsis. Chapter by chapter and paragraph by paragraph the facts are presented in a clear, non-speculative manner. In spite of that, the text reads easily. The book is complete and up-to-date; the bibliography is well chosen; the illustrations are excellent save for the colored photomicrographs.

Several splendid books on pathology and its branches have been published in recent years; they show a healthy tendency to interpret gross and microscopic findings and correlate them with clinical observations. This synopsis is written the old descriptive way. The appearance of a second edition within four years is indicative of the need for this book. It should be helpful as last reading before an examination and as a reference book to the practicing physician.